The hemalbumin was made as follows. The 3:5 DMSO solution (2.7 ml) is pipetted into a small beaker and a 300 µl volume of 1N NaOH (1:10 dilution) was added. This solvent was then added to the small beaker containing 231 mg of heme. This was stirred until the heme dissolved. The solubilized heme was then added by a syringe to a vial containing 100ml of 25% human serum albumin. The albumin vial was mixed by a vortex mixing during the heme addition. The amber, crystal-clear solution was stored at 4° C.

The hemalbumin acts by binding and/or converting nitric oxide to nitrates or nitrites and, therefore, inhibiting the vasodilatory effects of nitric oxide. The effects of hemalbumin in a dog treated with endotoxin is detailed in FIGS. **3**A–**3**C. Endotoxin was administered at 200 µg/kg as an i.v. infusion over 20 minutes. After administration of the endotoxin was complete, the animal developed progressive hypotension which is noted by a decline in the mean arterial blood pressure detailed in FIG. **3**A. The administration of hemalbumin (25 g of albumin in 50 cc) (2nd arrow) abrogated the fall in blood pressure due to endotoxin and stabilized the blood pressure. This stabilization of blood pressure was not observed with the administration of free albumin. FIGS. **3**B and **3**C respectively describe endotoxin (LPS) and hemalbumin effects on cardiac output and systemic vascular resistance.

Various references are cited above, the disclosures of which are incorporated in pertinent part by reference herein for the reasons cited.

The invention described and claimed herein is not to be limited in scope by the specific embodiments disclosed, since these embodiments are intended as illustrations of several aspects of the invention. Equivalent embodiments are intended to be within the scope of this invention. Indeed various modifications of the invention in addition to those shown and described herein will become apparent to those skilled in the art from the foregoing description. Such modifications are also intended to fall within the scope of the appended claims.

A variety of other hemoproteins or hemopeptides besides those specifically described herein are usable. The only requirement is that they effectively bind and/or catalyze nitric oxide oxidation. The methodology of Example 2 may be used to determine specific workable hemoproteins. Likewise, transition metals other than iron may be complexed in heme (metallohemoprotein) and analogously tested to determine effective complexes for nitric oxide removal. With respect to other transition metals and rationale for their NO binding activity, see Cotten, F. A., and Wilkinson, G., ADVANCED INORGANIC CHEMISTRY, A COMPREHENSIVE TEXT. Interscience Publishers, John Wiley and Sons, Inc., 1972, Chapter 22, "Complexes of π-Acceptor (π-Acid) Ligands, pages 682–727. Of course, any pharmaceutically acceptable salt forms of the hemoproteins may also be used as desired.

What is claimed is:

**1**. A method for treating a deleterious physiological effect in an animal caused by nitric oxide synthesis induced by a biological response modifier, the method comprising administering a therapeutically effective amount of a transition metal-hemoprotein selected from the group consisting of myoglobin, hemalbumin or methemalbumin to said animal.

**2**. A method for treatment of nitric oxide-induced hypotension comprising administering a therapeutically effective amount of a metallohemoprotein binding nitric oxide or catalyzing nitric oxide oxidation selected from the group consisting of myoglobin, hemalbumin or methemalbumin.

**3**. The method of claim **1** or **2** wherein the amount is from 0.1 to 10 g/kg body weight.

**4**. The method of claim **1** wherein the administering is intravascular.

**5**. The method of claim **1** wherein the deleterious physiological effect is systemic hypotension.

**6**. A method for treatment of an animal for systemic hypotension caused by nitric oxide comprising administering a therapeutically effective amount of myoglobin, hemalbumin or methemalbumin.

**7**. The method of claim **6** wherein the nitric oxide is induced by a cytokine.

**8**. The method according to claim **7** wherein the cytokine is tumor necrosis factor, interleukin-1, or interleukin-2.

**9**. A method for the treatment of an animal for systemic hypotension induced by endotoxin comprising administering a therapeutically effective amount of myoglobin, hemalbumin or methemalbumin.

**10**. A method for the treatment of septic shock in a patient comprising administering a therapeutically effective amount of a transition metal hemoprotein selected from the group consisting of myoglobin, hemalbumin or methemalbumin.

**11**. A method for the treatment of septic shock in a patient comprising administering a therapeutically effective amount of hemalbumin.

**12**. A method for treatment of systemic hypotension in a patient caused by nitric oxide production induced by a cytokine selected from the group consisting of tumor necrosis factor, interleukin-1, and interleukin-2 comprising administering an amount of methemalbumin or hemalbumin sufficient to bind or oxidize substantially all nitric oxide produced.

**13**. The method of claim **6**, **9** or **12** wherein the amount is from 0.1 to 10 g/kg body weight.

**14**. The method of claim **6**, **9** or **12** wherein the administering is intravascular.

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